The primary outcome was the progression of stenosis in symptomatic vessels at 6 months. Magnetic resonance findings were used to classify stenosis into 1 of 5 grades. Progression was defined as worsening of stenosis by 1 or more grades and regression of stenosis was defined as improvement of stenosis by 1 or more grades compared to the initial MRA study. Patients were also assessed with transcranial Doppler examinations.

From February 2000 to July 2003, 135 patients were enrolled in the trial. Sixty-seven were randomly assigned to cilostazol, and 68 were assigned to receive placebo. All patients in both groups also received 100 mg of aspirin daily.

Baseline patient characteristics and the location of intercranial lesions resulting in symptoms were similar in the two groups. During follow-up, there were no strokes or transient ischemic attacks, and two participants in each group had an acute coronary event. There was one unexplained death in each group. There were 20 dropouts in the cilostazol group (29.9%) and 14 dropouts in the placebo group (20.6%).

Progression of a symptomatic intercranial arterial stenosis occurred in three (6.7%) of the 45 patients available for follow up in the cilostazol group, and 15 (28.8%) of the 52 patients available for follow up in the placebo group. Regression of the intercranial lesion producing symptoms was noted in 24.4% of the cilostazol group and 15.4% of the placebo group. Overall progression of an intercranial arterial stenosis associated with symptoms was less frequent in the cilostazol group than the placebo group ($P = .008$). Transcranial Doppler studies also indicated that progression was less frequent in the cilostazol group than in the placebo group ($P = .001$). The progression rates of intercranial arterial lesions that were not associated with symptoms were not different between the two groups ($P = .384$).

Comment: Although this is a study of intracranial arterial lesions, the study is of interest in that it documents an effect of cilostazol on an established arterial stenosis associated with neurologic symptoms. The high frequency of progression and regression of intracranial lesions noted in this study suggests that intracranial arterial stenosis may behave much differently than extracranial carotid stenosis. Nevertheless, the idea that it may be possible to induce a relative high rate of regression of an arterial lesion by using pharmacologic manipulation is of considerable interest.

Clinical predictors of transient ischemic attack, stroke, or death within 30 days of carotid angioplasty and stenting

Kastrup A, Groschel K, Schulz JB, et al. Stroke 2005;36:787-91.

Conclusion: Patients with increased age, recent stroke, or hemispheric transient ischemic attack (TIA) have a higher risk of periprocedural complication after carotid angioplasty and stenting (CAS) for internal carotid artery (ICA) stenosis than younger patients or those with asymptomatic ICA stenosis or retinal TIA.

Summary: It appears combined stroke and death rates after CAS are higher in symptomatic than asymptomatic patients (Stroke 2003;34:813-819). The authors sought to identify *specific* clinical risk factors that may influence postprocedural complications after CAS. This was a retrospective review of a prospectively maintained database involving 299 patients (217 men, 82 women, mean age 69 ± 9 years) who underwent CAS for either asymptomatic (n = 129, 43%) or symptomatic (n = 170, 57%) ICA stenosis. Logistic regression analysis was used to determine risk factors associated with periprocedural (30-day) complications (any TIA, minor stroke, major stroke or death).

Overall, the 30-day TIA rate was 3.7%. The minor stroke rate was 5.3%, and the major stroke rate was 0.7%, with a death rate of 0.7%. Patients presenting with a minor stroke or hemispheric TIA had a higher risk of complication than asymptomatic patients (odds ratio [OR] 5.69; 95% confidence interval [CI], 2.03 to 19.57; $P < .001$). The complication rate in patients presenting with a retinal TIA and in asymptomatic patients were similar (OR, 1.42; 95% CI, 0.13 to 9.02; $P = .6$). Advanced age (OR 1.06; 95% CI 1 to 1.11; $P < .05$), stroke (OR, 8.0; 95% CI, 2.26 to 24.1; $P < .01$), or hemispheric TIA (OR 4.7; 95% CI, 1.16 to 13.3; $P = .004$) were all independent predictors of combined 30-day occurrence of any TIA, stroke, or death.

Comment: The identification of potential risk factors for CAS is crucial to proper patient selection for CAS and for patient counseling. This study helps identify presenting clinical features that may be important in selecting the patients for CAS. Unfortunately, angiographic features of the carotid stenoses were not reported. It is likely that a combination of angiographic and clinical features will ultimately prove most useful in the selection of patients for CAS.

Vascular access stenosis: comparison of arteriovenous grafts and fistulas

Maya ID, Oser R, Saddekni S, et al. Am J Kid Dis 2004;44:859-65.

Conclusion: Clinical evaluation for stenosis affecting a renal dialysis access is better for grafts than fistulas. Technical success and patency after angioplasty for stenoses in fistulas and grafts are essentially equal. After elective angioplasty, residual stenosis, high postprocedure access pressures, and female sex predict shorter access patency.

Summary: The authors studied clinical factors predictive of patency of a renal dialysis access after angioplasty. Relative outcomes of angioplasty of fistulas versus grafts were compared. During a 2-year period, all patients referred for a fistulogram for suspected access stenosis were studied. By protocol, angioplasty was performed for stenoses ≥50%. Each access was evaluated for degree of stenosis and number and location of stenotic lesions. In addition, a ratio of access to systemic systolic pressure was calculated. Multivariable analysis was used to evaluate clinical factors affecting access patency after angioplasty, and all procedures were tracked to calculate intervention-free access survival.

There were 543 fistulograms obtained during the study. Of these, 185 were obtained in fistulas and 358 in grafts. The likelihood of finding a significant stenosis was significantly higher in grafts than fistulas (69.75% vs 39.4%; $P < .001$). If a stenosis was present, patients with grafts were more likely to have two or more stenotic lesions (33.1% vs 12.5%; $P < .001$). After angioplasty, access to systemic pressure ratios and degree of stenosis were similar in fistulas and in grafts. In addition, intervention-free survival for grafts and fistulas was similar (6.2 months vs 7.5 months; $P = .36$). Residual access stenosis, female sex, and increased postangioplasty access pressure predicted diminished access survival ($P = .006$).

Comment: It is clear fistulas require fewer interventions than grafts to maintain patency. The current data, however, suggests that once angioplasty is required, subsequent patency is no better for fistulas than for grafts. Once stenosis develops in a dialysis access, the prognosis for intervention-free access survival is poor whether the access is synthetic or native.

Randomized controlled trial of prophylactic repair of hemodialysis arteriovenous graft stenosis

Dember LM, Holmberg EF, Kaufman JS. Kidney Int 2004;66:390-8.

Conclusion: Monitoring dialysis access grafts with static venous pressure monitoring and repairing identified stenoses prophylactically does not result in prolonged graft survival compared with a strategy of observation and repair of access only in the case of thrombosis.

Summary: Nonrandomized studies, often using historic controls, have suggested that prophylactic intervention for identified stenoses in dialysis access grafts will result in a reduction of overall access-related complications and access-related costs. Widespread recommendations therefore exist for access graft monitoring and prophylactic intervention to prevent thrombosis. The authors tested this theory in a prospective randomized trial at their institution. They compared prophylactic repair of arteriovenous graft stenoses with repair at the time of thrombosis.

Dialysis access patients were followed with measurements of static venous pressure. Static venous pressure is determined by turning off the dialysis blood pump and using the dialysis machine pressure displays to assess interaccess venous pressure. The static venous pressure ratio relates the intergraft pressure normalized to systolic blood pressure. The authors had previously defined an elevated static venous pressure ratio as >0.4. This had been determined to have the best predictive value for determining access stenosis (Kidney Int 1995;47:1364-73).

In this study, monthly static venous pressure ratios (SVPR) were determined. Patients randomized to the intervention group underwent angiography and angiographic or surgical repair of identified stenosis when the monthly SVPR was elevated (≥0.4). Patients randomized to the observation group only underwent stenosis repair in the case of access thrombosis or access clinical dysfunction.

The primary end point of the study was access abandonment. Access abandonment occurred in 14 patients in both the intervention group and the observation group during the 3.5 years of the study. Time to access abandonment also did not differ significantly between the treatment groups (hazard ratio for randomization to intervention, 1.75; 95% confidence interval [CI], 0.80 to 3.82; $P = 0.16$). The proportion of patients with a thrombotic event was greater in the observation group (72%) than in the intervention group (44%) ($P = .04$). Overall, thrombosis rates were similar in the two groups, however.

Radiologic or surgical procedures to repair stenosis or restore patency occurred at rates of 3.1 per patient year and 1.1 per patient year in the intervention and observation groups respectively (rate ratio, 2.75; 95% CI, 1.93 to 3.96; $P < .001$). Among patients whose access was abandoned because of thrombosis, the mean number of procedures before abandonment was 4.7 in the intervention group and 2.7 in the observation group.

Comment: This paper fails to confirm a relationship between access survival and access monitoring with prophylactic intervention for identified stenoses. The findings appear to be in direct conflict with results of previously nonrandomized studies. This study was small and occurred at a single institution. In addition, the authors' method of monitoring for access graft stenosis is not universally utilized. The paper does suggest the need for a larger, multicentered trial evaluating the merits of prophylactic dialysis access intervention. Recommendations for prophylactic repair of stenoses identified in dialysis access grafts may need to be reconsidered.